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Cis-regulatory module mediated control of sheath blight infection in Rice (*Oryza sativa* L.)

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ABSTRACT

Plant defense responses are orchestrated at the transcriptional level through *cis*-regulatory elements (CREs) organized as *cis*-regulatory modules (CRMs). This study systematically dissected the CRM architecture of resistance (R) gene promoters in two sheath blight-resistant rice genotypes, Tetep and Pankaj, to unravel the transcriptional logic underlying defense against *Rhizoctonia solani*. RNA-seq analysis identified 3,421 and 1,287 upregulated genes in Tetep and Pankaj, respectively, from which 119 canonical R-genes were annotated. *De novo* motif discovery using the MEME suite revealed six statistically enriched motifs (E-value < 1e-5) within 1 kb promoter sequences of R-genes. TOMTOM annotations mapped these motifs to three major transcription factor families: *DOF* (*DNA-binding with One Finger*), *AP2/ERF (APETALA2/Ethylene Response Factor*), and *RA1* (*RAMOSA1*)-like *C2H2* zinc fingers. Motif enrichment analysis via FIMO identified 3,371 highly significant occurrences across the promoter dataset, with *DOF* motifs being most abundant (n=1,690), followed by *C2H2* (n=739) and *AP2/ERF* motifs (n=484). CRM detection, defined as clustering of e"2 distinct TF family motifs within 300 bp windows, revealed combinatorial regulatory architectures in 168 R-gene promoters. Critically, 55 genes exhibited *DOF-ERF* co-occurrence, 65 genes showed *DOF-RA1* co-localization, and 11 genes harbored all three TF families, suggesting synergistic transcriptional control. *In silico* validation against functionally characterized genes confirmed biological relevance; notably, *Os09g0533600* (*BSR1*, a validated broad-spectrum resistance kinase) displayed dense CRM clustering. Several CRM-bearing R-genes co-localized with known quantitative trait loci (QTLs) for sheath blight resistance, including *qSBR-11-1, qSB-4, qSBR-7*, and *qSB-9*. These findings demonstrate that combinatorial CRM architecture, integrating *DOF*-mediated stress responses, *AP2/ERF* hormone signaling, and *RA1*-like regulatory innovation, orchestrates R-gene transcriptional activation during pathogen challenge. This work provides the comprehensive CRM catalog for rice disease resistance and establishes a framework for promoter engineering and breeding strategies aimed at enhancing quantitative and durable resistance to sheath blight.

Keywords: *AP2/ERF, Cis-regulatory modules, DOF, RAMOSA1, Resistance genes, Sheath blight, Tran scription factors*

Plant immunity relies on sophisticated transcriptional networks that coordinate defense gene expressions in response to pathogen attack. At the core of these networks are *cis*-regulatory elements (CREs), short DNA sequences in promoter regions that serve as recognition sites for transcription factors (TFs). When multiple CREs cluster together, they form *cis*-regulatory modules (CRMs) that function as integrated control units, enabling plants to mount precise and context-specific defense responses (Priya *et al.,* 2022). Resistance (R) genes represent the frontline of plant immunity, encoding proteins that detect pathogens and trigger defense signaling cascades. While extensive research has characterized their structural diversity and evolutionary patterns, emerging evidence reveals that R gene effectiveness depends critically on promoter-level regulation (Molla *et al.,* 2020). The spatial organization and activity of CREs within R gene promoters directly influence the timing, intensity, and tissue specificity of immune responses, making CRM architecture a key determinant of disease resistance.

Rice production faces significant challenges from sheath blight disease, caused by the necrotrophic

fungus *Rhizoctonia solani* (*AG1-IA*). This devastating pathogen ranks second only to rice blast in terms of crop damage, causing yield losses of 10–30% under favorable conditions (Senapati *et al.,* 2022). The absence of complete genetic resistance in commercial rice varieties highlights the urgent need to understand the molecular mechanisms governing host defense responses, particularly at the transcriptional level.

Recent advances in computational biology have revolutionized our ability to identify and characterize regulatory elements on a genome-wide scale. *De novo* motif discovery algorithms, combined with high-throughput sequencing data, enable systematic mapping of transcription factor binding sites and CRM organization. These approaches provide unprecedented insights into the regulatory logic underlying stress-responsive gene expression.

Despite progress in understanding individual transcription factor families involved in plant defense, the combinatorial nature of CRM-mediated gene regulation remains poorly characterized, especially in the context of host-pathogen interactions. This study addresses this knowledge gap by systematically analyzing the *cis*-regulatory architecture of resistance gene promoters in sheath blight-resistant rice genotypes. Our findings reveal the combinatorial CRM frameworks that orchestrate R gene transcriptional control and establish a foundation for targeted breeding strategies aimed at enhancing disease resistance.

Materials and Methods

To provide a comprehensive framework for the discovery of *cis*-regulatory modules (CRMs) in rice defense genes, a multi-step computational pipeline was implemented, beginning with transcriptome data processing and culminating in CRM identification.

RNA-seq Data Preprocessing and Differential Expression Analysis

RNA-seq data from two sheath blight-inoculated tolerant rice genotypes, Tetep and Pankaj, were obtained from the Indian Institute of Rice Research (IIRR), Hyderabad. Raw reads were subjected to quality control where adapter sequences and low-quality bases were trimmed using Trimmomatic. The filtered high-quality reads were then mapped to the reference rice genome assembly IRGSP-1.0 using HISAT2. Quantification of gene-level expression counts was performed using featureCounts. Differentially expressed genes (DEGs) were identified by comparing infected and control samples using the iDEP pipeline, with a false discovery rate (FDR) threshold of 0.05 and a fold change of e”2.

Identification of Resistance (R) Genes and Promoter Sequence Retrieval

To extract resistance gene candidates from the DEG set, annotation was achieved through the Plant Resistance Gene Database (PRGdb 4.0), which specializes in curated classification of R genes based on conserved domains such as NBS-LRR, RLK, and RLP. The DEG sequences were further annotated using HMMER and UniProt to cross-verify gene identity and domain architecture. Genes matching canonical R-gene classes involved in pathogen recognition and defense signaling were shortlisted. For each shortlisted R gene, the promoter region was defined as the 1000 base pairs upstream of the transcription start site (TSS), consistent with prior studies on *cis*-regulatory analysis (Motte *et al.,* 2015). These promoter sequences were retrieved from the Rice Annotation Project Database (RAP-DB) for subsequent motif-based analyses.

Motif Discovery and Transcription Factor Annotation

De novo motif discovery within the promoter sequences of identified R genes was conducted using the MEME suite, allowing for the identification of statistically enriched motifs. The discovered motifs were then matched against known transcription factor binding profiles using TOMTOM, querying against the JASPAR database to assign putative TF families to the identified motifs. To map occurrences of these motifs across all promoters precisely, the Find Individual Motif Occurrences (FIMO) tool was applied with a significance cutoff of p-value < 1e-4. FIMO outputs were filtered to retain only hits with q-values below 0.05 to ensure statistical rigor and minimize false positives.

Cis-Regulatory Module (CRM) Detection

CRMs were operationally defined as clusters consisting of at least two distinct TF family motifs localized within a 300 bp sliding window along the

promoter sequence. To detect such modules, a customized in-house Python script was developed that integrates FIMO motif occurrence data, grouping motifs based on spatial proximity and TF family diversity. This pipeline generates non-redundant CRM annotations detailing their start/end coordinates, constituent TF families, and motif counts. Overlapping CRM windows were merged to consolidate clustered regulatory modules, thereby avoiding redundancy in CRM identification. The 300 bp window size was selected based on previous studies showing that functional CRM elements typically cluster within this distance (Priya *et al.,* 2022).

RESULTS AND DISCUSSION

Differential Expression and Resistance Gene Annotation

A comparative transcriptome profiling of two sheath blight-tolerant rice genotypes, Tetep and Pankaj, identified 3,421 and 1,287 significantly upregulated genes respectively (FDR < 0.05, fold change e" 2) upon *Rhizoctonia solani* infection. This substantial differential response underscores the extensive transcriptional reprogramming that occurs during pathogen recognition and defense activation in these resistant varieties. The marked difference in DEG numbers between Tetep and Pankaj (3,421 vs 1,287) suggests distinct resistance mechanisms or varying degrees of constitutive resistance between these genotypes.

Annotation of these DEGs for resistance (R) gene domains using UniProt and HMMER revealed 71 putative R-genes in Tetep, including 66 containing leucine-rich repeat (LRR) domains and 3 receptor-like kinases (RLKs). Conversely, Pankaj yielded 48 putative R-genes comprising 40 LRRs and a comparatively elevated proportion of 8 RLKs. The prominence of LRR-containing genes concurs with previous findings by Molla et al. (2020), who reported induction of 52 NLR/LRR genes specifically in Tetep during sheath blight stress. The higher RLK representation in Pankaj (16.7% vs 4.2% in Tetep) is particularly noteworthy, as RLKs have been implicated in early pathogen perception and MAPK cascade activation in rice (Xiao *et al.,* 2021).

Motif Discovery in Promoter Regions of R-Genes

To decipher the regulatory architecture underlying resistance (R) gene activation, 1 kb promoter regions upstream of candidate R genes from both Tetep and Pankaj were subjected to de novo motif discovery using the MEME suite. This approach yielded six highly significant motifs, each identified across multiple promoter sites with strong statistical support (E-value < 1e-5). The sequence logos visualized the most conserved base positions for each motif, with the prominence of certain nucleotides indicating likely transcription factor binding sites within the R gene promoters (Fig 1).

The first motif (motif 1: width 35, 121 sites, E-value 1.4e-95) is characterized by a prominent AT-rich core, which often corresponds to binding sites for *DOF* transcription factors, known mediators of plant defense. Motif 3, featuring a recurring GAGAGAG repeat (width 21, 67 sites), may signal binding preferences for *C2H2*-type zinc finger proteins, such as the *RAMOSA1* (*RA1*)-like class, consistent with regulatory modules involved in defense signaling. Motif 5 (width 29, 45 sites) is distinctly GC-

Fig 1. Sequence logos for each discovered motif, representing the nucleotide composition and positional entropy at every site and motif width, instance count, and enrichment statistics.

rich, closely matching the canonical GCC-box—a key recognition site for *AP2/ERF* transcription factors, which orchestrate ethylene and jasmonic acid signaling pathways during plant immune responses.

Motifs 2 and 4, with widths of 50 and 47 nucleotides and distinct arrangements of AT and GC content, suggest binding sites for additional regulatory factors, potentially linked to high-order combinatorial control of gene expression. Finally, motif 6, an AT-rich sequence of width 32 (14 sites), may represent novel or uncharacterized *cis*-elements that recur in R gene promoters and require further investigation. The collective enrichment of these motifs points to a complex combinatorial operating unit in the transcriptional control of disease resistance genes in rice.

Motif Annotation and enrichment

To annotate the *de novo* motifs identified by MEME, we combined the promoter motifs from both resistant genotypes (Tetep and Pankaj) into a single motif file and queried them against two curated reference collections using TOMTOM: the JASPAR non-redundant plant motif set (802 motifs) and the *Arabidopsis* DAP-seq database (872 motifs). This analysis resulted in 103 significant matches against JASPAR and 194 matches against the *Arabidopsis* collection (q-value < 0.05), providing reliable TF family-level assignments for a substantial proportion of MEME-derived motifs.

Representative TOMTOM alignments (Table 1) illustrated the robustness of these associations. The AT-richmotif (DTNHWWTTTTTTTTTWH WTTTWTYTWWWTWTWWTT) aligned with *DOF3.6*, a member of the *DOF* (DNA-binding with one finger) transcription factor family. In rice, *DOF* TFs have been reported to mediate defense-related transcriptional reprogramming, often acting as integrators of biotic stress signals (Xian *et al.,* 2020). The presence of MEME-1 in R-gene promoters suggests that *DOF*-mediated regulation may enhance transcriptional activation during pathogen challenge. This interpretation is further supported by recent studies showing that *DOF* proteins function as both activators and repressors through direct DNA interaction, enabling dynamic regulation of defense gene expression (Yanagisawa, 2002).

The GC-rich MEME-5 motif (GSRGCGGCGGCSVSKSSGGCGBSGBKGGG) displayed high similarity to the GCC-box element, which is the canonical binding site for *AP2/ERF* transcription factors. The GCC-box is well established as a central *cis*-element in defense signaling, particularly in rice responses to necrotrophic pathogens and ethylene/jasmonic acid pathways (Gu *et al.,* 2017). The enrichment of *AP2/ERF* motifs in R-gene promoters corroborates the central role of this superfamily in stress-responsive transcriptional networks, with approximately 170 *AP2/ERF* genes identified in the rice genome (Rashid *et al.,* 2012).

The AG-rich degenerate motif (DGRGRGAGAGAGRGGAGGRGR) (MEME-3) corresponded to *RAMOSA1* (*RA1*), a *C2H2* zinc finger transcription factor. *C2H2*-type TFs in rice have been implicated in transcriptional control of defense-related genes. Their ability to bind degenerate AG-rich motifs aligns with the AG-repeat structure of MEME-3, suggesting a role in modulating chromatin accessibility or fine-tuning defense gene expression. The identification of *RA1*-like motifs in R-gene promoters is particularly intriguing given that *RA1* is absent in rice, wheat, and oats; however, its *cis*-regulatory logic appears conserved through *RA1*-like *C2H2* zinc finger proteins that may substitute functionally in stress contexts. Comparable patterns have been reported by Yokotani et al. (2013), who observed dense motif clustering in the promoters of resistant rice cultivars during *R. solani* infection.

In contrast, three motifs (RWTKKATCWT CKAACAMHAATAAAAAWACTAA,CTAATYA TRGAYTAATTAGGCTTAAAARATTYGTCTCG YGAWTTACM and YGAGACGAATCTTTTGAG CCTAATTAATCCATSATTAGHMHATGTGRKKY) did not yield significant or functionally interpretable hits in the TOMTOM searches. These were retained as "unknown motifs" for reporting. Nevertheless,

Table 1. List of discovered motifs along with their q-values and annotation details

Discovered Motif	q-value	Annotation
DGRGRGAGAGAGRGGAGGRGR	0.0001422	*DOF3.6*
DTNHWWTTTTTTTTTWHWTTTWTYTWWWTWTWWTT	2.92E-06	*ERF9*
GSRGCGGCGGCSVSKSSGGCGBSGBKGGG	1.80E-06	*GRMZM2G003927_P01*

their recurrent presence in R-gene promoters suggests potential novel *cis*-elements that could be discovered with expanded motif libraries or experimental validation in future studies.

Motif Enrichment in R-gene Promoters

To assess the biological significance of discovered promoter motifs, we mapped the six MEME-identified motifs across all R gene promoters using FIMO, applying a stringent enrichment threshold of $p < 0.0001$. This analysis yielded an extensive 3,371 motif occurrences distributed across the promoter dataset, indicating both high motif recurrence and statistical confidence in motif-DNA matches. Specifically, *DOF* motifs were most abundant (n = 1,690), followed by C2H2 zinc finger motifs (n = 739), *AP2/ERF* motifs (n = 484), and uncharacterized motifs (n = 458).

The high frequency and broad distribution of these motif hits underscore their likely functional involvement in gene regulatory logic. Prior studies in rice and other cereals have established that clustered or recurrent binding motifs, especially AT-rich and GC-rich elements such as *DOF*-binding sites and the classical GCC-box, are strongly indicative of combinatorial promoter activity often found at immune-relevant loci and central to stress-inducible transcriptional programs. The predominance of *DOF* motifs (50.2% of total occurrences) is particularly striking and suggests these plant-specific TFs may play an underappreciated role in defense gene regulation. This finding extends beyond their traditionally recognized functions in development and carbohydrate metabolism to include pathogen-responsive transcriptional control.

Critically, the mapping of motif instances at the genome scale using FIMO enables quantitative prioritization of specific promoters enriched for multiple, diverse motif types. In our dataset, motif instances belonging to *DOF*, *AP2/ER*F, and *RA1*-like *C2H2* zinc finger families were abundant; these patterns mirror recent literature describing how defense genes, and especially R genes, in rice and other crops are governed by complex *cis*-regulatory modules harboring multiple TF recognition sites.

CRM identification

The custom in-house pipeline for *cis*-regulatory module (CRM) detection produced multiple CRM clusters in the promoter regions of R genes in rice. The analysis identified CRMs as clustered occurrences of two or more distinct TF family motifs co-localized within 300 bp promoter windows. This window size balances sensitivity for detecting co-regulation while minimizing false positives from randomly dispersed motifs. The analysis of motif occurrences across 168 unique resistance (R) gene promoter sequences revealed distinct CRM clusters, each grouped according to their constituent transcription factor (TF) families. Among these, 120 genes were dominated by the *DOF3.6* motif, typically associated with *LRR* and *RLK* genes. The *ERF9* motif was found in 40 gene promoters, while motifs belonging to the *RA1* family were observed in 30 genes. Importantly, 55 genes possessed both *DOF* and *ERF* binding domains, and 65 genes contained both *DOF* and *RA1* motif sites within their promoters. Notably, 11 genes were identified with all three binding domains (*DOF, ERF*, and *RA1*), and 19 genes displayed the presence of all four binding domains (*DOF, ERF*, *RA1*, and *unknown*).

This combinatorial CRM architecture suggests a sophisticated transcriptional regulatory network where multiple TF families converge to orchestrate R-gene expression. The high degree of co-occurrence (55 genes with *DOF-ERF*, 65 with *DOF-RA1* combinations) indicates that combinatorial regulation is not exceptional but rather a common feature of defense gene promoters. This modular architecture likely provides regulatory robustness ensuring appropriate gene activation even if individual TFs are compromised and enables fine-tuning of expression levels through TF dosage effects or competitive binding.

Key Candidate Genes and CRM Architecture

Several R genes emerged as prominent CRM carriers, with dense clusters involving *DOF*, *ERF*, and *RAMOSA1* motifs. *Os09g0533600*, corresponding to the broad-spectrum resistance kinase *BSR1* (RLCK), harbored multiple CRMs combining *DOF3.6, ERF9*, and *RAMOSA1* motifs (Fig 2). *BSR1* has been extensively validated through overexpression and CRISPR/Cas9 genome editing studies enhancing rice resistance against multiple pathogens, confirming its pivotal role in immune signaling pathways and validating the motif-based regulatory predictions (Matsushita *et al.,* 2016). The

Table 2. List of key R genes identified in the study along with their CRM compositions.

Gene ID	CRM composition (TF families)
Os01g0140400	*DOF + ERF*
Os02g0136900	*DOF + ERF*
Os05g0125200	*DOF + ERF*
Os09g0533600	*DOF + ERF*
Os11g0308800	*DOF + ERF*
Os04g0649700	*DOF + RA1*
Os01g0769700	*DOF + RA1*
Os10g0468500	*DOF + RA1*
Os07g0542400	*ERF + RA1*
Os03g0211900	*ERF + RA1*
Os11g0232100	*DOF + ERF + RA1 + Unknown*
Os06g0638500	*DOF + ERF + RA1 + Unknown*

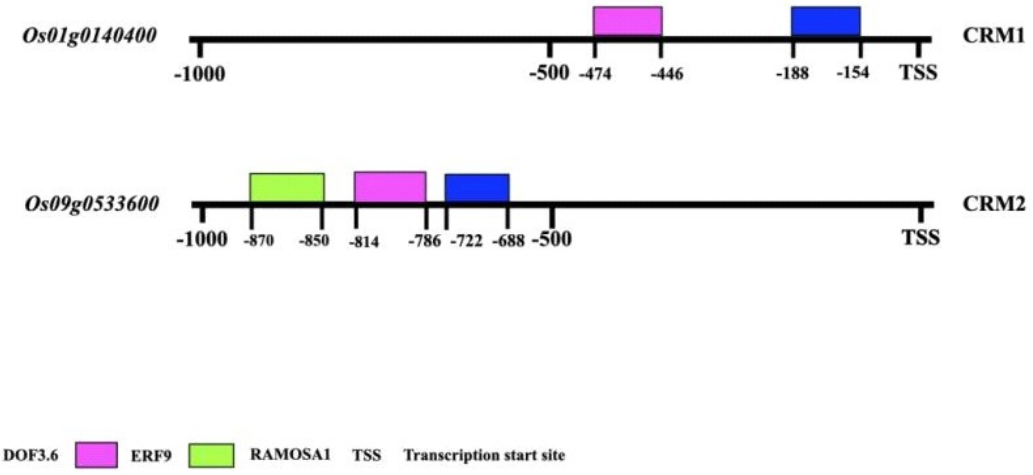

Fig 2. *Cis*-regulatory modules (CRMs) and their motif organization in the 1 kb promoters of *Os01g0140400* (CRM1) and *Os09g0533600* (CRM2). Each CRM window is shown with its constituent-colored motif bars—*DOF3.6* (blue), *ERF9* (magenta), and *RAMOSA1* (green)—indicating their position and span within the CRM.

co-occurrence of three distinct TF binding motifs in the *BSR1* promoter suggests that its broad-spectrum activity may stem from its ability to integrate multiple transcriptional inputs, enabling responsive expression across diverse pathogen contexts.

Another candidate, *Os05g0125200*, annotated as a lectin receptor-like kinase (*LecRK*), exhibited CRMs comprising *DOF3.6* and *ERF9* motifs. *LecRK* family members have established roles in modulating *MAPK* signaling and insect/pathogen resistance in rice and *Arabidopsis* (Xiao *et al.,* 2021). The CRM enrichment supports the transcriptional regulation of LecRK genes as vital components of the immune response network. The *DOF-ERF* combination in the *LecRK* promoter suggests coordinated regulation through independent but synergistic pathways: DOF-mediated general stress responsiveness and *ERF*-mediated hormone-dependent defense activation.

In this study, we identified 12 key CRM-bearing R genes displaying combinatorial promoter motifs involving *AP2/ERF, DOF* and *RA1* TF families, which likely underlie sheath blight resistance variation between rice cultivars (Table 2). Several of these genes co-localize with known quantitative trait loci (QTLs) for sheath blight resistance, providing strong genetic evidence for their involvement in disease resistance. *Os01g0140400* (from the *DOF3.6* and

ERF9 CRM group) overlaps with *qSBR-11-1*, a major QTL on chromosome 11 strongly associated with sheath blight resistance. Similarly, *Os02g0136900* is co-localizing with the reported *qSB-4* QTL for sheath blight resistance (Li *et al.,* 2019). *Os07g0542400*, possessing a distinctive *AP2/ERF-DOF-RA1* motif architecture, resides in the *qSBR-7* region defined by Chen et al. (2018). *Os09g0533600* (BSR1), already validated in transgenic and genome editing functional assays, aligns with the qSB-9 QTL on chromosome 9 strongly implicated in sheath blight resistance (Maeda *et al.,* 2024). *Os11g0308800* is co-localized with *qSBR-11-3*, one of the key loci identified by Matsumoto et al. (2022). The gene *Os06g0638500* lies within the *qSB-6* locus on chromosome 6 involved in sheath blight resistance (Kushwaha *et al.,* 2020). Similarly, *Os01g0769700*, precisely associated with *qSB-1* on chromosome 1 (Patel *et al.,* 2021), *Os03g0211900* located at *qSB-3* on chromosome 3 (Singh *et al.,* 2020), and *Os04g0649700*, mapped to *qSB-4*, are also related to sheath blight resistance (Li *et al.,* 2019). Notably, *Os01g0140400*, *Os06g0638500*, and *Os11g0308800* show significant differential expression in resistant and susceptible rice cultivars during sheath blight infection, as reported by Yang et al. (2022) and Wang et al. (2022).

This CRM architecture facilitates the identification of promoter regions under potential combinatorial regulation, influencing plant defense against pathogens. In our analysis, majority of R gene promoters contained at least one CRM instance, and many carried densely packed arrays of three or more motif families. These findings suggest that coordinated action by several TF types—*DOF*s, *AP2/ERF*s, and *C2H2*—may drive robust and finely tuned activation of R gene expression following pathogen recognition. The modular nature of these CRMs implies that resistance could be enhanced not only through R gene overexpression but also through strategic promoter engineering to optimize CRM configuration and composition.

CONCLUSION

This study employed an integrative computational pipeline combining transcriptome profiling, *de novo* promoter motif discovery, and *cis*-regulatory module (CRM) identification to unravel the regulatory landscape governing disease resistance genes in rice. Our analysis revealed statistically significant enrichment of motifs recognized by key transcription factor families—*DOF, AP2/ERF* and *RAMOSA1*—which frequently co-occurred within tight CRMs, suggesting combinatorial regulation of R gene expression. The identification of CRMs across promoters of known and novel R genes confirms established regulatory frameworks while uncovering previously uncharacterized modules involved in rice sheath blight defense. Validation against functionally characterized genes such as *BSR1* underscores the biological relevance of our motif-based predictions, while detection of less explored candidates like lectin receptor kinases highlights the discovery potential of this approach. Our findings demonstrate that effective plant immunity relies on intricate transcriptional regulatory networks coordinated through modular *cis*-elements. The CRM catalog developed herein provides a foundation for understanding the regulatory logic underlying rice disease resistance and offers a framework for targeted breeding strategies aimed at enhancing quantitative and durable resistance to sheath blight.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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